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Letters to the Editor

Case-finding and case-holding in Malawi

Sir,

Following correspondence with the Director of LEPRA concerning some of my early impressions of leprosy control in the Balaka area, I thought it might be of value to record some thoughts on the two subjects of case-finding and case-holding in this part of Malawi. Case finding would, I am sure be a great deal easier if the population at large and prospective patients in particular, (a) are aware of the simple ways of suspecting leprosy, (b) know where to go for confirmation or otherwise of their suspicions as well as (c) obtain subsequent medical help if needed (d) without a significant measure of social ostracism. It is the person who voluntarily comes forward for help that by and large turns out to be the maximally motivated and co-operative patient. To facilitate this situation inhibitive factors physical and especially social, must be at a minimum in the society.

Case holding also depends on the aforegoing factors but my short experience has shown that the leprosy workers role is here vital. The project grassroot planning must be geared to making case holding easy for the patient. The clinics should be timed regularly, be situated at physically convenient points for the patients, held on days socially convenient to the patient. The leprosy worker should endeavour to develop harmonious medical relationship with his patients. From the time of admission to the time of release from control there is to be continued imploring and coaxing of the patient vis-á-vis his receiving – let alone swallowing – treatment.

As part of health education the patient should be made to understand the main objectives of a control project namely (1) to prevent people from getting leprosy by destroying the primary causative agent, i.e. the leprosy bacillus, (2) to minimize the discomfort of leprosy in those patients already with disease. In endeavouring to accomplish these tasks the patient has an important role to play by ensuring that he co-operates with the leprosy worker and thereby ensuring that his load of germs is destroyed and that progress of his disease is arrested, while discomforts therefrom are alleviated.

We are lucky in this country because ostracisim resulting from leprosy is

of little significance. Among the elderly the ritual of attending the clinic to get the 'magic pill' is something to look forward to. Some of the elderly and middle-aged are most reluctant to be discharged, regardless of the degree of incapacity brought about by the leprosy. Case-holding among adolescents is more difficult. Some of those without obvious leprosy do not want to be known to have leprosy in case this prejudices their chances of marriage (important in agrarian and subsistence communities) and employment. Case holding among children of course is related to the degree of responsibility of their parents or guardians.

On the subject of children and the value of schools examinations, which some observers have found to be highly significant, I have learnt one important point already, and that is that a low incidence in schoolchildren may be misleading if only a small proportion of all children are able to find a place in schools. This situation is often linked with poverty, a factor which is almost certainly one of the many predisposing factors in the spread of leprosy.

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Does clofazimine have any value in the management of reversal reaction?

Sir,

With reference to the article on clofazimine in *Leprosy Review* **50**, 1979, 134–44, I would like to make the following comments:

Two papers (references 1 and 2 below) are frequently quoted in support of the contention that clofazimine is effective in the management of reversal reactions, but neither paper really makes the case.

In her paper Schulz[2] writes: 'In our patients neuritis was adequately controlled after an average of 3½ months of treatment with clofazimine.' I submit that an average time lapse of 3½ months before the control of neuritis is in reality evidence of the ineffectiveness of clofazimine in these cases.

Both Pfaltzgraff and Schulz deserve thanks and congratulations for having controlled after an average of 3½ months of treatment with clofazimine.' I submit that average time lapse of 3½ months before the control of neuritis is in reality evidence of the ineffectiveness of clofazimine in these cases.

Both Pfalzgraff and Schulze deserve thanks and congratulations for having tackled a problem most of us have evaded, but surely it is time to settle the issue one way or the other. At least we should stop recommending clofazimine for the management of reversal reaction until more definite evidence is available.

Barnetson, *et al.*[3] have done leprosy patients a great service by exploding

the myth of dapsone as a causative agent in reversal reactions by a controlled clinical trial. It is my belief that the alleged effectiveness of clofazimine in reversal reactions is also a myth. Have any of your esteemed readers evidence to the contrary?

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2 Schulz J. 44 months' experience in the treatment of leprosy with clofazimine. *Lepr Rev,* 1971, **42**, 178.

3 Barnetson R StC, Pearson JMH, Rees RJW. Evidence for prevention of borderline leprosy reactions by dapsone. *Lancet,* 1976, **11**, 1171.

Leprosy control in the Southern Province of Zambia

Sir,

In April 1978 we introduced a modification of the History Record card used by LEPRA in Malawi as a pilot project in our area of leprosy control in the Southern Province of Zambia, which is based on this Hospital. This area includes three districts (Mazabuka, Monze and Gwembe) and serves a population of approximately 200,000 people. We used the card to record standard information on 100 consecutive patients with previously undiagnosed leprosy. Whilst fully acknowledging that no statistical conclusions can be drawn from such a small sample, we nevertheless feel that some of the results are worth recording. Of the 100 patients studied

(1) the majority had had their disease for longer than one year before presentation
(2) 17% presented with inactive disease, 10 borderline, 6 tuberculoid and 1 indeterminate,
(3) 26% were in a state of Type 1 (*syn.* reversal, upgrading) reaction on first presentation. For many, the appearance of oedema or nerve damage was a real factor leading to presentation at Chikankata. Most of these had severe reactions of recent onset and they responded well to therapy. (This finding is in keeping with experience in Ethiopia and suggests that dapsone therapy is not a major factor in the causation of this type of reaction in leprosy.),

(4) 52% had some form of nerve deficit and 42% had actual deformity in at least one limb or eye at the time of diagnosis and finally,
(5) no fewer than 36% of patients presenting for diagnosis had positive smears.

In view of the considerable amount of work that has gone into leprosy control in this area for at least two decades and previously noted by Gauntlett (1969)[1] and du Plessis (1977),[2] the number of patients presenting with positive smears, and the high incidence of established deformity in patients presenting for the first time, is disconcerting.

We are collecting further data, but feel that the evidence to date suggests that there may be much more leprosy in this area than has been so far diagnosed, especially as most patients were self-reporting.

The main purpose of this letter is to suggest that evaluation by small sample surveys, carried out by independent observers, may be of increasing importance in areas such as our own, where considerable resources have been invested attempting to control leprosy, often over long periods of time, and where it is essential to find out what progress has been made.

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[1] Gauntlett SL. Leprosy control in the Southern Province of Zambia. *Lepr Rev,* 1969, **40,** 223–32.
[2] du Plessis PA. Unpublished observations, 1977.

Dapsone and Nerve Damage

Dear Sir,

In the course of my travels over a wide area of the Far East, my attention has been drawn to a considerable number of cases in which the use of dapsone, particularly in high doses from the outset, has been associated, in the opinion of a number of experienced observers, with adverse reactions and nerve damage. I have also been asked if dapsone may, itself, cause nerve damage after long-term use. I am, therefore, prompted to write about three aspects of the use of this valuable drug, namely: 1. the correct dose in relation to body

weight; 2. the possibility that the long-term use of dapsone in some patients may be damaging to nerves; and 3. the possible relation between the use of dapsone in doses in the order of 50–100 mg. daily, from the outset in all cases, and the development of nerve damage.

1. DAPSONE IN RELATION TO BODY WEIGHT

The WHO Export Committee on Leprosy, Fifth Report, Technical Report Series 607, 1977, clearly states that dapsone should be used in doses of 50–100 mg daily in full size adults, on a basis of 6–10 mg/kg body weight per week, with correspondingly smaller dosages for children. I feel that I should draw attention to the fact that in many areas of Asia, the average adult weighs only 45–55 kg – and often suffers from anaemia and not infrequently, from intestinal parasites or other diseases. I therefore wish to record a word of warning about the routine use of 100 mg daily in small patients in this part of the world when 50 mg daily would conform with the recommended doseage.

2. DOES THE LONG-TERM USE OF DAPSONE CONTRIBUTE TO NERVE DAMAGE?

Whilst fully appreciating that lepromatous patients must take dapsone, or some anti-leprosy drug on a very long-term basis (after a period of dual therapy), I feel that I should record results of discussions with a number of clinicians working in East Asia who feel that dapsone, itself, may play some part in the production of chronic neurological problems. I am further stimulated to put forward these views on hearing that some Indian leprologists, at their recent conference in Madras, have suggested that the long-term effects of dapsone in this context should be further investigated. Particularly in view of a number of well-documented reports in the literature of peripheral neuritis in dermatological patients on treatment with dapsone for conditions such as *dermatitis herpetiformis,* I wonder if there is any conclusive scientific work which indicates that dapsone as a drug may, in fact, be neuropathogenic? Many patients from all sectors of the immunological spectrum have been seen with chronic neural complaints and/or slowly progressive nerve deficits, in whom symptons disappear on cessation of dapsone and substitution of another antileprotic. Whilst active disease with dapsone resistance may need to be considered in some more recent patients, this is not likely to be a common causative factor. Is it possible that dapsone's long continued use may contribute to eventual nerve damage in addition to the fibrosis which, understandably, follows bacillary invasion and infiltration of nerves by the leprosy infection? Are there medications in common useage that may increase or decrease peripheral nerve damage in leprosy? Are we, in fact, increasing disability by therapy?

3. A POSSIBLE RELATIONSHIP BETWEEN THE USE OF DAPSONE IN DOSES OF 50–100 MG DAILY FROM THE OUTSET, AND NERVE DAMAGE

This is a matter of much greater and more urgent concern than the above. It was stated, both in Mexico and in the *Lancet* of 17 November 1976, by Barnetson, Pearson and Rees, that higher doses of dapsone may actually prevent reversal reactions. This is contrary to the clinical impressions of many of us who can cite numbers of patients in whom the sudden introduction of such levels of dapsone, even after the use of other antileprotics, has apparently precipitated acute nerve lesions, usually as part of a reversal reaction, and a more chronic type of neural discomfort in some lepromatous type patients. These events are often polyneuritic and leave permanent neural deficits, though I realise that with adequate, early corticosteroid therapy, one may expect a degree of nerve recovery in many cases. However, the use of steroids in the field, under conditions as I seem them in this large area, is far from straightforward. Whilst their use after an acute neural deficit may result in an apparently complete response, we cannot be sure how long such improvement will be maintained, and we must remember the limitations of such therapy. First, in many control programmes there is already difficulty in gaining co-operation of the patients and the general public; if a new diagnosed patient develops paralysis soon after commencing treatment this could hamper the whole programme. This is not theory – it has happened in several areas and usually there is a considerable period of delay before further progress with the control programme is made. Second, the corticosteroids ideally should start at once; certainly within six weeks of the development of the deficit if there is to be real hope of significant recovery of nerve function. In many areas patients can only be seen once in three or even six months – and it is impractical to tell them to 'Hurry to clinic' if a reaction occurs.

Thirdly, it has been suggested that Paramedical Workers should be given supplies of corticosteroids to treat patients with acute nerve deficits and other reaction problems. This is not acceptable to some governments – and it would certainly add complications to the treatment regimes. Simplicity of treatment is one of the reasons given for suggesting that all patients, irrespective of leprosy type, should receive the same dose of dapsone. In some countries, corticosteroids are easily obtained on the local market and have already created many problems in the management of patients. Once a patient has been given corticosteroids for a short time he often states that they make him 'feel good' and so he goes and buys more himself and it is not uncommon to see these patients become dependent on corticosteroids. One young woman has been taking 30 mg prednisolone a day for 3 years to control ENL, and a taxi driver was taking 15–20 mg prednisolone daily for about a year to control the lesions of BL-type leprosy, but neither took any antileprotics! Corticosteroids in the right hands can help – but given without adequate supervision and

understanding can produce more problems than they solve; at least, amongst the very unstable borderline group of patients that are so common in East Asia.

Some experienced workers suggest that with the use of antileprotics other than dapsone, e.g. Lamprene Thiacetasone and Thiambutasone, the incidence of acute neural deficit may be less and there may, possibly, be a decrease in the amount of permanent nerve damage. Some of these drugs may be less effective antileprotics, but the immunologically unstable patients who may benefit from their use are also those in whom secondary drug resistance is less likely to be a problem.

At the Mexico conference, Dr Waters, speaking from the chair, after the paper on Reversal Reaction, pointed out that there may well be definite racial variations in leprosy that, at present, have been little investigated. He has worked with a number of racial groups. I am very conscious of racial variations, and with this possibility in mind, I appeal to readers to try to run their own investigations; treating patients carefully and keeping complete records so that they, or someone else, will be able, eventually, to assess what treatment is best for that particular group of patients. Please do not just accept recommendations based on work in another country without carefully observing the effects in your own group.

Nerve damage is the most disabling physical result of leprosy. Are we increasing or decreasing it by our present therapy?

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